TECHNICAL REVIEW COMMENTS ON ARKWOOD INC. SITE RISK ASSESSMENT

General Comments

This report will require substantial revision. The comments which follow outline specific reasons for this, and include some specific suggestions and recommendations which may be employed. Also, please add an Executive Summary to the report. Portions of Section 7 in its current form might be used as a basis for this.

Chapter 1 (Introduction)

Section 1.2.3, Page 1-5: Please revise the last sentence to reflect listing of the site on the NPL.

section 1.8, Page 1-18: Certain information regarding analytical methodology used in the RI needs to be provided in order to evaluate this document. More specifically: 1) The detection limits, and their relation to contaminant levels which would be of concern, need to be provided; 2) It should be specified whether the reported soil/sediment concentrations were based on wet or dry soil/sediment weight, and whether the aqueous contaminant concentrations were based on filtered or unfiltered water samples.

Section 1.8, Page 1-19: Please delete Bullet #9 and the final sentence of Bullet #5.

Chapter 2 (Methodology)

Section 2.2.2, Bullet \$4: The derivation of benzo[a]pyrene equivalents as described cannot be accepted at this time. Please refer to additional comments on this subject which are included under Chapter 3.

Section 2.2.2: Please indicate the manner in which non-detects were factored into the determination of exposure point concentrations.

Section 2.2.2, Bullet 45: Total PNAs should be used to evaluate non-carcinogenic effects of this chemical class, as both carcinogenic and noncarcinogenic PAHs could presumably elicit systemic toxic effects. Please refer to additional comments on this subject which are included under Chapter 7.

Bection 2.2.3, Page 2-5: This section describes comparison to ARARs, but no such comparisons were located in the report. Please add this to the report, and include comparison with to-beconsidered criteria (e.g., Drinking Water Health Advisories, Ambient Water Quality Criteria, MCLGs,) which are available for some of these contaminants.

section 2.2.4, Page 2-5: The most recent Agency position regarding future use scenarios may be located in the Federal Register (Federal Register 53, No. 245 (12/21/88), pg. 51425). In brief, this indicates that a "reasonable maximum exposure scenario" (as opposed to either "most probable" or "worst-case improbable" scenarios) is to be addressed in the baseline risk assessment. The exposure scenarios provided may be acceptable, however, some modification of assumptions might be required for the purpose of developing site clean-up goals.

Section 2.3, Page 2-7: Typo in the definition of "C" under Inhalation, should be mg/cu.m., not mg/cu.mg.

Section 2.3, Page 2-8: Soil and water are described as the only media employed to compute chemical intakes. Please add text describing the manner in which other media of potential concern (e.g., air, ingestion of biota) were evaluated.

Section 2.5.1, Page 2-11: Acceptable intakes for chronic and subchronic exposures were apparently developed for this document. This might be acceptable, however, the methodology employed for such derivations would have to be described, and toxicity values derived by EPA would generally take precedence. Please refer to additional comments under Chapter 7 regarding documentation of toxicity values in this report.

Chapter 3 (Indicator Compounds)

Section 3.1, Page 3-2: The treatment of all carcinogenic PAHs as equivalent to benzo[a]pyrene (BaP) for risk assessment purposes may be overly conservative. However, the use of relative potency factors for estimating PAH carcinogenic potencies is not yet EPA-approved (though it is my understanding that both the general approach and some relative potency estimates are undergoing Agency review). In the absence of Agency approval, it should be assumed for purposes of this report that the carcinogenic PAHs are equipotent with BaP, in accordance with available Agency guidance (e.g., Superfund Public Health Evaluation Manual, Health Effects Assessment for Polycyclic Aromatic Hydrocarbons).

Also, does the final indicator list represent "all the detected constituents" as mentioned in Section 2.1 (at the bottom of Page 2-2)? If not, please add text indicating the manner in which these selections were made.

Chapter 4 (Exposure Point Concentrations)

Section 4.2.3.2, Page 4-3: The final paragraph claims that the 1.01~mg/l is not exceeded. The data shows 1.0~-~2.3~ppm in the spring. Please explain this inconsistency.

Section 4.2.3.5, Page 4-4: There may be other affected surface waters and sediments which need consideration (e.g., a stained sediment which was observed along the roadside ditch to the northwest of the site).

Section 4.3, Page 4-7: Please provide support for the assertion that the concentration data collected in the Arkwood site RI are log-normally distributed. This subject is also mentioned in Section 2.2.2.

Section 4.3, Page 4-7: EPA is not convinced that maximum concentrations should not be employed in this risk assessment. These concentrations may be more appropriate than representative concentrations for estimating subchronic intakes, particularly in the event of occasional, intermittent exposure to "hotspots." Further, calculation of a subchronic intake for assumed intermittent exposures to maximum concentrations may generate an artificially low intake estimate for use in non-carcinogenic risk evaluation (see additional comments on this subject under Chapter 5). This issue may best be addressed by a screening procedure, whereby intakes based on a single exposure event and calculated using maximum concentrations could be generated, and then compared to AIS values. The relation of these (i.e., the hazard index) could generate a rationale to dismiss the issue, or might suggest a second, less conservative screening step. In this fashion, maximum concentration data could be employed to assist in risk characterization for the site.

Please see additional comments on subchronic intakes under Chapters 5 and 7.

Section 4.4 (applies to section as a whole): Please clarify the distinction between the New Cricket Spring, and its headwaters. In particular, the potential of either the Spring or its headwaters to be used as an intentional (as opposed to incidental) drinking water source should be discussed, and revisions made to the exposure assumptions if necessary.

Also, please indicate how the exposure frequencies (e.g., six exposures per year to railroad ditch soils as described in Section 4.4.1.1; monthly visitation by a hiker as described in Section 4.4.2) employed in this report were selected.

section 4.4.2.1, Part A: EPA does not agree with the 0.5 "soil exposure factor." The reasons are as follows. This factor appears based on a stated assumption that "half of the soil to which an individual will be exposed will be unaffected soil", and on an implicit assumption that a visitor will randomly contact various site areas. EPA does not interpret the RI as supportive of this. In contrast, most of the site appears contaminated, and areas exhibiting the highest contaminant concentrations appear to be the most accessible and attractive. Further, distribution of site contaminants may already be accounted for in the derivation of representative concentrations.

Part B: It is unclear to the reviewers whether the 0.5 factor mentioned here represents the same one as described in Part A. The text needs clarification.

Section 4.4.3, Page 4-11: Please tone down the first sentence of this section.

Section 4.4.3.1: The chosen soil ingestion values are consistent with Agency guidance (see attached OSWER Directive), but do not represent pica behavior. Please delete the parenthetical reference to pica.

Sections 4.5: It may be inferred from this summary (as well as Sections 4.4.2.3 and 4.4.3.2) that the Respondents consider the railroad ditch to be inaccessible to persons other than railroad company employees. EPA does not agree with this assertion. Please modify the risk assessment to include exposures to railroad ditch contaminants by persons other than railroad company employees.

section 4.5, Exposure Scenario III: For New Cricket Spring, the dismissal of dermal absorption based on insignificance relative to ingestion is inconsistent with its inclusion in Scenario II. Also, please correct the text of Section 4.4.3.2, which incorrectly indicates identical exposure assumptions for New Cricket Spring in Scenarios II and III.

Section 4.5, Tables 4-2 and 4-3: The indication in Table 4-2 that surface water is analyzed is inconsistent with text in Section 4.4.1.2 which suggests otherwise. An apparent typo was located in Table 4-3; the first of two "Private Wells On-Site" referrals under the Exposure Point column should seemingly be "Private Wells Off-Site."

Chapter 5 (Chemical Intakes)

General Comment: Neither the chemical intakes, nor the calculations by which they were derived, are considered to be adequately documented in this draft. Description of the manner by which subchronic intakes were calculated was particularly deficient. Chronic and subchronic intakes should be reported separately for each contaminant, at each exposure point. This information should be presented in a manner which allows the specific exposure pathway and assumptions employed in their derivation to be readily identifiable. This information should be reported in this Chapter, as opposed to the Appendix.

The manner in which intermittent and/or subchronic intakes were developed for use in non-carcinogenic risk estimation is not well described. It should be noted that normalization of intermittent intakes based on frequency of exposure (e.g., given 6 events per year, using a 6/365 correction to generate an intake estimate for non-carcinogenicity evaluation) can be inappropriate. Please indicate in the revised report the manner in which this issue is being addressed.

Section 5.3 (and Table 5-2): It appears that a single, 0.5 factor was applied to both PCP and PNAs to account for absorption across gut or dermal barriers, in the presence or absence of soils. Such an approach seems overly simplistic, and may particularly overstate the relevance of dermal absorption.

For the oral exposure route, no absorption factor should be applied, as GI tract absorption is already accounted for in development of cancer potency factors or RfDs for oral exposure. A retardation factor to account for the presence of soil, however, can be appropriate. The 0.5 factor might be appropriately conservative for this purpose, as the affected soils are reportedly of relatively low organic carbon content. For the same reason, this 0.5 factor might be applied to dioxins as well, as opposed to the 0.3 factor reported in Kimlrough et. al. An acceptable approach for calculation of dermal in akes from aqueous solution is provided in the Appendices of the Superfund Exposure Assessment Manual (EPA/540/1-88/001). Lesser EPA guidance is available for calculating dermal intake of contaminants from soil matrices. The 0.01 value reported in Kimbrough et al. 1984, might be employed for this purpose.

Table 5-2: The 16 kg figure for weight of children aged 6-12 seems low. Please support or revise.

Tables 5-3 and 5-4: The values employed for Y and T, particularly for childhood or subchronic exposures, need to be provided in each instance where these equations are used to developed chemical intakes. Also, the Table 5-3B for oral intake contains a dermal absorption factor.

Chapter 6 (Toxicity Profiles)

General Comment: Information provided in these toxicity profiles should be referenced. The Respondents should also add text which indicates that toxicity profiles were not provided for each indicator, and the reason for this. A Toxicological Effects section, which indicates that this compound is a suspected human carcinogen, should be added to the profile for benzo[a]pyrene.

Derivation of Clean-Up Level, Page 6-19: The argument presented in support of a 258 mg/kg PCP clean-up level is unconvincing. It seems to be based on a comparison of the detected PCP concentration (5.7 mg/l) with the reported PCP solubility of 14 mg/l, and an inference that this 0.41 ratio may then be used as a correction factor for deriving PCP target levels. The Agency is not convinced of the validity of this approach. In any event, this is a subject which is more relevant to the FS. Therefore, please delete this discussion (i.e., delete Pages 6-18 thru 6-21) from the endangerment assessment.

Proposal for PCP Clean-Up Levels, Page 6-20: Bullet #3 does not necessarily support the proposal, since relative toxicity (as well as relative concentration) pertains to this issue. Bullet #8 is based on the use of relative potency factors for PNA-C, which is unacceptable at this time.

Proposal for PCP Clean-Up Levels, Page 6-21: The purpose of a baseline risk assessment is to document risks which may be posed by a site in the absence of remediation. The text on Page 6-21 beginning with and following Conclusion #4 is viewed as premature and inappropriate for inclusion in a baseline risk assessment; please delete it.

Chapter 7 (Risk Characterization)

General comment: This Risk Characterization is not adequately developed (though sections of it might be used in an Executive Summary). A major problem is that it does not adequately document the manner in which the risk estimates were derived. Rather, it simply presents (in Tables 7-1 and 7-2) the summarized results. Please expand this chapter to document the manner in which a cancer risk estimate or hazard index was developed from each of the individual chemical intakes derived in the report. Further, the only toxicity criteria which were located in this report were the AIS and AIC values for PCP. All toxicity criteria (AIS, AIC, RfDs, CPPs) used in this risk assessment should be specifically reported and referenced. Should toxicity criteria derived by ERM-Southwest be employed in the risk assessment, their derivations should be documented as well.

Please refer to Chapter 7 of the Superfund Public Health Evaluation Manual (SPHEM) for guidance regarding the manner in whic: HI and cancer risk estimates for individual contaminants are 13 be summed to account for potential additivity of multiple contaminants and pathways. This approach should be employed in this risk assessment.

Thapter 8 of the SPHEM describes a manner by which preliminary target levels may be derived for inclusion in a risk assessment. This process can be simplified by use of the same methodologies and assumptions employed in the risk characterization, and by simply generating for now the concentration levels corresponding to a 1E-6 risk or HI value of 1 (i.e., by not attempting apportionment at this time, as these are preliminary goals only).

Page 7-1, first paragraph: The methodology used to calculate intakes is provided in Section 5. The intakes were not; they are listed in the Appendix.

Page 7-1, third paragraph: The derivation of TEFs used in this risk assessment for dioxins/furans needs to be documented.

Page 7-1, final paragraph: Please qualify the first sentence to read, "...without expected adverse effects."

Page 7-2, fourth paragraph: EPA has published an oral AIC for naphthalene of 4E-1 mg/kg/day, in the 1986 Health and Environmental Effects Profile for this chemical (EPA 600/X-86/241). This should be used to develop HI values for naphthalene in the Arkwood risk assessment. This AIC may also be used to generate a toxicity value to which total PAHs may be compared for screening purposes, which is preferable to no evaluation at all. Such a toxicity value should incorporate a safety factor (i.e., use 4E-3 mg/kg/day), since naphtalene is relatively non-toxic and is structurally quite different from larger PAHs.

Similarly, The potential for dioxins/furans to elicit non-carcinogenic effects needs to be addressed. A RfD for dioxin of 0.000001 ug/kg/day was developed by EPA (Drinking Water Criteria Document for 2,3,7,8-TCDD. EPA 600/X-84-194-I, also cited in the ATSDR Toxicological Profile for this compound). Using this as an AIC, HI estimates can be derived.

Page 7-2, last sentence: This sentence is viewed as inappropriately definitive. "This methodology is generally considered to overstate the true risk, perhaps by an order of magnitude or more" conveys the point more appropriately.

Tables 7-1 and 7-2: The distinction in exposure scenarios and assumptions for children aged 2-6 from those aged 6-12 was not located elsewhere in the document. Why do these values not differ in Table 7-1? Table 7-2 should report non-carcinogenic hazard indices for total PNAs as well (see previous comment).

Assumptions/Uncertainties, Page 7-5: Some balance ought to be added to the discussion of assumptions. There are numerous assumptions made in most any risk assessment, including this one, which may understate risk. Examples include: chemicals not identified or lacking toxicity criteria were not quantitated, pica behavior was not considered, and synergistic effects due to simultaneous exposure to chemical mixtures may occur. Such assumptions should be identified and included in this discussion.

Page 7-6: The second bullet is too definitive, "... is considered likely to overstate human risk." would be more appropriate.

Chapter 8 (Summary and Conclusions)

General Comment: The summary and conclusions should represent an unbiased interpretation of the risk assessment. Some of these summary/conclusion statements included here do not reflect such interpretation, and therefore, require some qualification and/or additions. They should reflect pertinent results for carcinogenic and noncarcinogenic risk posed by all site indicators. Pending results of the revised risk assessment, the following revisions may be acceptable. For Section 8.1:

Bullet #1 (and #3 of Final Conclusions): Replace "There is no..." with "There is no evidence to date of..."

Bullet #2a: Use, "No carcinogenic PNAs were detected in the railroad ditch."

Bullet #6: Mention the risk estimated due to carcinogenic PNAs, and results of the HI evaluation.

Section 8.2: The final conclusions in the revised risk assessment should reflect a balanced interpretation. Please delete the text from Bullet #4 which implies that Scenario III is unrealistic.

Bibliography

The reference to USEPA, 1986b, mentioned in Section 2.5.1, was not located in the Bibliography, nor was a 1986a. Please check the Bibliograph section for completeness.

<u>Appendix</u>

We attempted to reproduce some of the intake values in these tables, and were unable to do so. This is viewed to support the above comments which indicate a need to improve the documentation of the manner in which these estimates were derived.

In addition, Table A-2 indicates that intakes generated from dermal exposure are less than those for oral exposure. This is inconsistent with typical results of risk assessments, and likely is attributable to the assumptions employed for dermal absorption.